DISCUSSION

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Among the most disabling, and costly, chronic diseases affecting Americans (as well as innumerable other persons) is osteoarthritis (Kraus and Stoddard, 1989). The degradation of cartilage, the clinical sign of osteoarthritis, has motivated the search for understanding the mechanisms of normal and pathological synovial joint tribology. The cause of this disease is unknown, in part because it takes so long to develop—a decade or more likely passes before the aneural cartilage has fissured to the point where bone contact instigates pain. That mechanical factors may contribute to the initiation or progression of cartilage destruction (Sokoloff, 1969) is suggested both by the high loads the joints support and the absence in cartilage of the usual channels of physiological communication—cartilage tissue has neither blood vessels nor nerves. Thus, cartilage cells (chondrocytes) may depend upon the mechanical environment to control their function as well as to transport nutrients and metabolites (Hall et al., 1988; Madreperla et al., 1985; Sah et al., 1989).

Quantifying the relevant physical parameters in the joint, i.e., cartilage stress, fluid pressure and flow, energy dissipation via friction and consequent temperature rise, appears central to enhanced understanding.

Thus, it is amazing, no tragic, that no scientific agreement exists on even the macroscopic questions—how does cartilage sustain the repeated loadings of a lifetime for those fortunate to maintain painless mobility, and perform this task with surprisingly low friction. A standard text quotes two prominent researchers in cartilage biomechanics: “Evidence to support the various hypotheses (hydrodynamic, elastohydrodynamic, boundary, squeeze-film—as well as unique schemes—‘weeping’ and ‘boosted’) has often been indirect and conjectural.” (Mow and Mak, 1987). Ateshian et al. continues this controversy.

Early attempts to apply well-known tribological theories—hydrodynamic and exclusively boundary layer—have been abandoned, given the reciprocating, low-velocity motion and long stationary periods of joint loading and the measured whole joint friction coefficients (0.002 to 0.02). Based on physical experiments with rubber foam against glass, McCutchen (1959) put forth the theory of “weeping lubrication.” In the normal physiological state the collagen-hydrophilic proteoglycan matrix of cartilage is saturated with fluid, some 80 percent by weight. The bone side of the one-to-two mm. thick layer is impermeable, so when load is applied, the incompressible fluid in it will be expressed to the low-pressure synovial cavity through one path tangentially in the layer, many mm. long, and a second radial path, a mm. or so long, to and thence through the interarticular gap, the “weeping” component of flow. McCutchen (1975) estimated the partitioning of fluid between these paths.

More recently in Macirowski et al. (1994), cited by Ateshian et al., experimental data defining the detailed three-dimensional geometry of in situ human hip cartilage and the pressure distributions on the hip socket as the cartilage was loaded were applied in a finite-element model of the layer to calculate the partitioning of the fluid between the paths, the cartilage matrix stresses, and the fluid pressures in the gap and deep in the cartilage. The average matrix stresses were low, tenths of a MPa, not surprising since when discs of cartilage are loaded via a porous platen which permits expression of the interstitial fluid, the drained modulus of the human or bovine tissue network is measured to be less than 1 MPa (Lee et al., 1981). However, pressures on acetabula cartilage in vivo are in excess of several MPa, typically 5 MPa during the single-leg support phase of normal gait, becoming as high as 18 MPa during rising from a low chair and when descending steps (Hodge et al., 1986, Hodge et al., 1989). Given the low modulus of the matrix, such measured pressures must be that of the constrained fluid in the cartilage and in the interarticular gap.

In the 1970s a group at Rensselaer Polytechnic Institute (later at Columbia) modeled cartilage biomechanics, first with Torzilli-Mow theory and then with biphasic KLM theory, the formulation adopted by Ateshian et al. With this background I address my problems with the Ateshian et al. article in three overlapping areas, analysis, experiment and recognition of other contributors to the field.

The history of joint lubrication research given by Ateshian et al. leaves out some interesting parts. McCutchen’s 1959 article in Nature that proposed weeping lubrication was followed in the same issue by Lewis and McCutchen (1959) which showed that cartilage wept fluid when compressed. An elegant chemical-tracer experiment by Lewis measured the amount of fluid squeezed out and showed that it came from outside the cartilage cells. Lewis’s experiment deserved more notice than it has received.

In listing theories of joint lubrication Ateshian et al. omit the “mechanical pumping mechanism” and consequent flow of fluid out of and back into the cartilage proposed by Mansour et al. (1973) and Torzilli and Mow (1976a and b). Mechanical pumping was said to follow from Torzilli-Mow theory, which in turn was said to correct a purported, fundamental (but nonexistent) error in the consolidation theory of M. A. Biot (1955).

Later a somewhat similar flow pattern, the “biphasic self-generating mechanism” of Mow and Lai (1980) that Ateshian et al. mention, was predicted via the subsequent KLM model, but only by using a free-draining indenter pressed against the cartilage. No fluid pressure can be developed beneath a free-draining indenter.

The KLM model was presented as a “reformulation of the biphasic model for articular cartilage proposed by Torzilli and Mow . . .” (Mow et al., 1980). In truth, it was identical to Biot theory, a fact widely known and formally demonstrated by Simon in 1992. Ateshian et al. write that they use “the linear biphasic theory (Mow et al., 1980)” without explaining that it is a rederivation of Biot’s (1955) consolidation theory.

Nor do they mention that computations for an impervious indenter sliding over a layer of poroelastic material were done by Uzowire in his unpublished Ph.D. thesis at Rensselaer Polytechnic Institute in 1980. Uzowire found pore pressures nearly equal to the applied loading.

In “Model Formulation” Ateshian et al. write: “In the present friction model . . . no fluid film separates the surfaces. . . . This assumption is partly based on previous reports (as reviewed above) that EHL (elastohydrodynamic lubrication) predictions of fluid film thicknesses are generally smaller than the surface roughness of cartilage, and that typical predictions of squeeze-film times are on the order of seconds. . . . Hence . . . it is assumed in this model that any pre-existing fluid film has been significantly, if not entirely, depleted.”

Ateshian et al. seem to believe that the fluid film vanishes, or nearly so, once the high spots on opposing rubbing surfaces have touched. In fact it vanishes only at the contacting high spots. Elsewhere its thickness is of the order of the peak-to-valley roughness of the surfaces, a micron or so, (see, for example, McCutchen, 1980), or greater depending on the congruency of the joint, as demonstrated by the temporal changes in the pressure distribution in Macirowski (1994). Fluid flows through the resulting passages parallel to the interface and escapes from the loaded region. The escape of fluid lowers the pressure in the partial film to a value lower than that in the pores. Pore fluid flows into the partial film, and this “weeping” flow greatly reduces the rate at which the pressure in the film falls, likewise slowing the rise in the load supported by contacting high spots and the consequent rise in friction.

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No contradiction exists between this weeping flow and calculated flows into and out of squeeze films between porous surfaces (see for example Hou et al., 1992), because these calculations assume no contact between high spots on opposing surfaces.

Ateeshian et al. perpetuate a different confusion by calling cartilage viscoelastic instead of poroelastic. A poroelastic material responds sluggishly to changes in applied loading because the response involves flow of fluid through its pores. The time dependence response of a poroelastic material is influenced by the size and shape of the sample and the permeability of its boundaries; the time dependence of a viscoelastic material is not thus influenced.

The paper’s Eq. (16) revives McCutchen’s (1962) hydrostatic clamping effect. Raising the hydrostatic pressure in fluid in which a bearing is immersed was expected to raise its friction even if the bearing load remained unaltered. However in a simple experiment McCutchen (1978) found no obvious change in friction when the pressure in a bearing’s bathing fluid was raised to 100 atmospheres.

Ateeshian et al. find a rise in friction, but their experimental result (Fig. 4) is four times greater than predicted and lasts half as long. There is another possible explanation for it. Thanks to the escape path for fluid between the surface of the cartilage and the indenter there will, during the interval of increasing strain, be a thin layer of high stress and low pressure next to this surface, a milder version of the high-stress, low pressure layer at the filter end. During the stress relaxation at constant thickness the extra stress near the rubbing surface should diffuse quickly into the material below, causing a fall in the solid stress at the surface, and therefore the friction, that lasts until overbalanced by stress diffusing though the sample from the filter end.

Ateeshian et al. could pursue the hydrostatic clamping effect by putting their entire apparatus in a hyperbaric chamber, measuring the friction with the cartilage at equilibrium, and seeing if it rises when the chamber pressure is raised. A pressure of 0.22 MPa would match the peak calculated pressure in their experiment.

Supporting the cartilage layer on a permeable structure, as the authors do, shortens the experiment by allowing the pore fluid to escape easily. The authors might have noted the high pore pressure that they find early in the experiment would last much longer in a real joint as Macirowksi et al. (1994) has shown. Also their choice of a rotating device means whatever friction force they measure ranges from zero at the stationary center to a maximum at the periphery.

Finally, it is refreshing to have finally an opportunity to attempt to engage the Columbia group in a scientific discussion. This same issue of “no fluid film” arose in “On a theoretical solution for the frictionless rolling contact of cylindrical biphasic articular cartilage layers,” by Ateshian and Wang (1995), J. Biomech., 28 (11), 1341–1355, where they also ignored the flow of fluid in the contact area between the cylinders. A “Comment” raising this omission published by Mann and McCutchen in J. Biomech. (1997), 30 (1), 99 prompted Ateshian and Wang to reply with the single sentence: “We would like to thank the authors (myself and C. W. McCutchen) for their clarification concerning our statement on “The absence of fluid exudation...” Since then Ateeshian has published “A theoretical formulation for boundary friction in articular cartilage” (1997), ASME J. Biomech. Eng., 119, 81–86, which rediscovers McCutchen’s self-pressurized hydrostatic lubrication, but claims no “weeping” occurs. McCutchen has submitted a “Comment” to J. Biomech. Eng. noting that Ateeshian finds no “fluid exudation at the contact interface” because he assumes that no fluid escapes along the crack between the rubbing surfaces (as explained) in Ateeshian, G.A., Lai, W.M., Zhu, W. B. and Mow, V. C. (1994) “An asymptotic solution for the contact of two biphasic cartilage layers,” J. Biomech., 27, 1347–1360.

However someone else challenges the Columbia scientists, they have a ready reply. Since their model does not admit of a film, a film does not exist!

Additional References


Authors’ Closure

It is with interest that we read Professor Mann’s discussion of our paper and further elaborations on the history of cartilage biotribology research. Clearly, Professor Mann supports certain aspects of the “weeping” lubrication mechanism proposed by McCutchen (1959) which we do not necessarily agree with. However, in the introduction and discussion sections of our paper, we have already addressed Dr. McCutchen’s theory in relation to the mathematical model and corresponding experimental verifications which we present. We simply emphasize that our mathematical model demonstrates that low friction can be achieved in cartilage even when the fluid lubricant between the cartilage surfaces has been depleted. As such, unlike Dr. McCutchen’s theory, our proposed model does not appeal for a mechanism of fluid exudation into the spaces between contacting cartilage surface asperities to explain the low cartilage friction. Dr. McCutchen’s conjecture as to the fluid flow pathway has long been viewed with skepticism by other experts in the field of lubrication. For example, Professor Duncan Dowson indicated in 1973 that “The aspect of the [weeping] concept which appeared to worry many people when it was first proposed was the idea that fluid would